Conclusions: Although GPIIb/IIIa blockade in the absence of heparin may not significantly prolong the ACT, it appears to potentiate heparin effect in a dose dependent manner. The increment in ACT with c7E3 was proportional to the degree of underlying anticoagulation (as reflected by the baseline ACT), with a possible interaction at high heparin doses. Thus, increased bleeding with c7E3 may be avoidable if the heparin dose is lowered. The effect of c7E3 on TEG variables implies that ACT does not measure the time to onset of fibrin formation, but rather the time to attainment of a certain clot strength.

4:15

809-2 Intravenous Nitroglycerin Causing Heparin Resistance

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The existence of an interaction between nitroglycerin (TNG) and heparin (H) has remained controversial. To evaluate this interaction, Sprague Dawley rats (5 in each group, n = 50) were anesthetized and activated clotting time (ACT) determined before and following H. H, 70 units/kg bolus followed by 5 units/hr was given and ACT increased from 60 ± 11 to 135 ± 38 sec ($p < 0.01$), at 1 hour. ACT remained prolonged with H infusion for 4 hrs (135 ± 38 sec hr 1, 128 ± 25 sec hr 2, 139 ± 25 sec hr 3, and 155 ± 35 sec hr 4). TNG IV in a propylene glycol solution was administered 0.2 mg bolus followed by 0.1 mg/hr. H caused ACT to increase from 43 ± 6 to 100 ± 4 sec and then TNG caused ACT to decrease at 1, 2 and 3 hrs, 64 ± 11, 55 ± 12 and 53 ± 4 sec respectively. TNG without propylene glycol caused a similar attenuation of H effect. Both high (0.20 mg, and low (0.04 mg) boluses of TNG every 3 min attenuated ACT prolongation by H as well as shortening the time to attenuation of H effect. TNG (0.2 mg) decreased ACT from 183 ± 28 to 132 ± 12 5 min, 123 ± 10 10 min, and 119 ± 3 15 min; while with TNG (0.04 mg) ACT decreased from 138 ± 22 to 134 ± 2 at 5 min, 143 ± 2 15 min, 116 ± 22 20 min, and 92 ± 11 at 25 min. Saline in amounts equal to the volume of TNG was given over 3 hrs with no change in ACT. In this *in vivo* model TNG & H show a definite interaction with the prolongation in ACT caused by H decreased by concomitant administration of TNG.

4:30

809-3 Comparison Between Low Molecular Weight Heparin and Standard Heparin in Unstable Angina

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Standard heparin, by intravenous route (IVH), is often used in the treatment of patients with acute ischemic syndromes and reduces the number of ischemics events in unstable angina patients. However, its administration is hampered by the need for repeated aPTT measurements and dosage adjustments. We tested the hypothesis that the clinical outcome and safety of subcutaneous low molecular weight heparin (LMWH), which does not require repeated monitoring of aPTT, are similar to those of standard heparin in patients (P) with unstable angina. We also compared the pharmacological effect of subcutaneous standard heparin (SCH) with IVH. The number of revascularization (PTCA, CABG), hemorrhagic events (at 1 week), as well as aPTT at baseline, 6 hours post-bolus (aPTT1), and at 12 hours intervals thereafter (aPTT2–6) were obtained in 350 patients (90% Braunwald's Class III/B), mean age 67 ± 11 years. All received IVH 5000 IU as an initial bolus and 200 mg ASA daily. Seventy P (LMWH) received 15000 anti-Xa U, every 12 h; 110 P (SCH) 12500–17500 IU, every 12 h and 170 P (IVH) about 1200 IU/h. Only SCH and IVH dosages were adjusted by an algorithm to keep aPTT 1.5–3.0 times control. Results:

		LMWH	SCH	IVH	p
aPTT1 (post-bolus)	(s)	55 ± 23*	64 ± 40	98 ± 18	<0.0001
aPTT2-6 (mean)	(s)	40 ± 06*	80 ± 40	88 ± 38	0.093
Therapeutic Level	(%)	–	57.3	55.9	0.967
Supratherapeutic Level	(%)	–	21.8	24.1	0.607
Events (PTCA, CABG)	(%)	5.7	5.5	4.7	0.922
Hemorrhage	(%)	5.7	6.4	7.1	0.924

*Not compared in statistical analysis

Except for the first measured aPTT 6h post-bolus, therapeutic level can be achieved with subcutaneous administration of standard heparin as well as intravenous route. There was no difference in clinical outcome and hemorrhagic events between patients treated with IVH, SCH and LMWH. Thus, LMWH may be more practical and equally efficacious alternative to the use of IVH in unstable angina patients.

4:45

809-4 A Randomized Comparison of Higher and Lower Dose Aspirin on Collagen-Induced Platelet Aggregation

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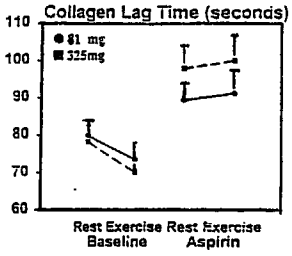
The optimal dosage of aspirin for protection against cardiovascular disease is uncertain, particularly in different clinical settings. Since heavy exertion has been shown to trigger myocardial infarction, and an increase in platelet aggregation with exercise has been reported, we compared the effects of two doses of aspirin (81 mg, n = 19 and 325 mg, n = 21) on platelet aggregability before and after treadmill exercise.

In a randomized, double-blind, parallel study of 40 healthy males, Born aggregometry was performed to determine the lag time to aggregation following exposure to 0.19 mg/ml collagen (CLT). A longer CLT indicates reduced responsiveness. Both 81 mg and 325 mg aspirin significantly increased the CLT compared to that observed pre-aspirin, however the 325 mg increased the CLT to a greater extent ($p < 0.05$). Prior to aspirin, exercise was associated with a shortening of CLT. This increase in response was abolished by both doses of aspirin.



Conclusion: While both 81 mg and 325 mg aspirin significantly inhibited platelet reactivity to collagen, a greater inhibition was observed with 325 mg. Since collagen is an important platelet agonist *in vivo*, the clinical significance of this dosage-related difference warrants further study.

810 Cardiopulmonary Resuscitation

Wednesday, March 27, 1996, 4:00 p.m.–5:00 p.m.
Orange County Convention Center, Room F1

4:00

810-1 Emergency Coronary Bypass or Angioplasty in Patients With Electromechanical Dissociation or Refractory Ventricular Fibrillation: Enhanced Survival With Left Ventricular Venting

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Left ventricular (LV) distension during ventricular fibrillation (VF) or electromechanical dissociation (EMD) can reduce subendocardial perfusion and thus impair LV performance. Despite the institution of percutaneous cardiopulmonary bypass support (PCPS) during cardiac arrest (CA), 25 pts remained in refractory VF (21 pts) or EMD (4 pts). All pts had complete loss of LV ejection during or shortly after coronary intervention (9 pts) or routine angiography (16 pts). Fourteen pts (Group 1) had percutaneous LV venting (8–10F pigtail catheter in LV) prior to emergency bypass surgery (N = 11) or after emergency angioplasty (N = 3) and outcomes were compared to 11 pts who went directly to bypass surgery and did not have LV venting (Group 2). Baseline clinical and angiographic characteristics were similar for both groups. Pts had PCPS established during VF or EMD with a mean time to PCPS of 18 ± 11 min (17 ± 14 min for Group 1 and 19 ± 8 min for Group 2). Group 1 had LV venting prior to surgery (N = 11) or after angioplasty (N = 3) and Group 2 was immediately transferred to surgery on PCPS without venting. Mean pulmonary artery pressure (MPAP) for both groups was (39 ± 4 mm Hg on PCPS, but MPAP dropped to 10 ± 4 mm Hg in Group 1 pts after LV venting was initiated ($p < 0.001$). Overall survival was 44%. Survival for Group 1 was 71% versus only 9% for Group 2 ($p < 0.007$). These data suggest that LV venting can decrease LV distension and may improve survival when used as an adjunct to PCPS during emergency bypass surgery or angioplasty in CA pts.